COMPARATIVE STUDY OF EFFECTIVENESS OF PERCUTANEOUS COLLAGEN INDUCTION THERAPY AND DERMABRASION IN ACNE SCARS

This dissertation is submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfilment of the requirement of the award for the degree of

M.D BRANCH XX

DERMATOLOGY, VENEREOLOGY AND LEPROSY

STANLEY MEDICAL COLLEGE

CHENNAI – 600 001

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CERTIFICATE

Certified that this dissertation entitled **"COMPARITIVE STUDY ON THE EFFECTIVENESS OF PERCUTANEOUS COLLAGEN INDUCTION THERAPY AND DERMABRASION IN ACNE SCARS"** is a bonafide work done by **Dr.S.NITHYA PRIYADHARSHINI,** post Graduate Student of the Department of Dermatology, Venerology and Leprosy, Stanley Medical College, Chennai – 600 001 during the academic Year 2010 – 2013. This work has not been submitted previously for the award of any degree.

Dr. V.ANANDAN, M.D.,
Professor and Head of Department,
Department of Dermatology & Leprology,
Stanley Medical College,
Chennai – 600001.

Prof. Dr S.GEETHALAKSHMI, M.D.,Ph. D
Dean
Stanley Medical College,
Chennai – 600001

DECLARATION

I solemnly declare that the dissertation titled, **COMPARATIVE STUDY OF EFFECTIVENESS OF PERCUTANEOUS COLLAGEN INDUCTION THERAPY AND DERMABRASION IN ACNE SCARS** was done by me at **Stanley Medical College and Hospital during 2010-2013** under the guidance and supervision of my **Chief Dr. V. Anandan, M.D.**

The dissertation is submitted to **THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY** towards the partial fulfilment of requirement for the award of **M.D.Degree (Branch XX) in DERMATOLOGY, VENEREOLOGY & LEPRSOY.**

Place:

Date:

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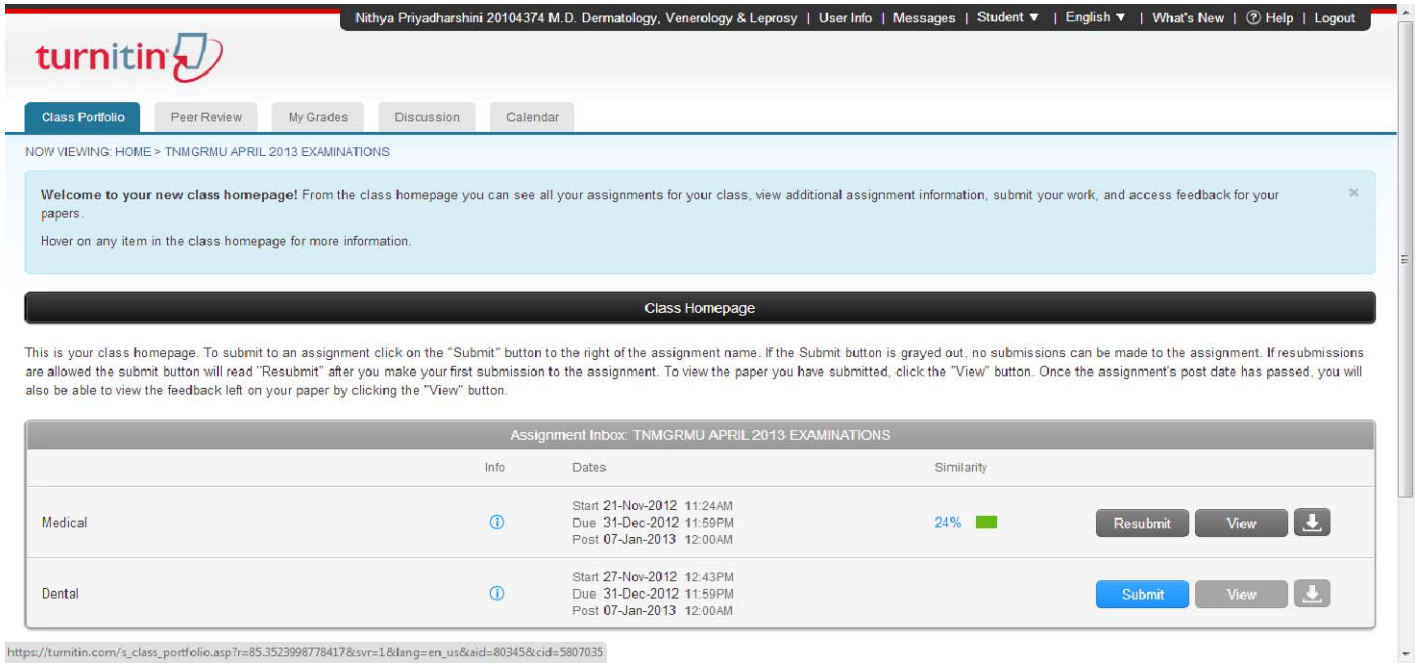
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CONTENTS

INTRODUCTION

Acne is a self limiting disease of pilosebaceous unit seen predominantly in adolescence, clinically characterized by papules, pustules, comedones, nodules and cysts and may result in scarring.

A scar is a process of healing of tissue after injury. Acne scars occurs due to compromised collagen production as the wound heals, resulting in topographical depressions. Scarring occurs early and may affect 95% of patients with acne depending on severity and delay in treatment. A variety of modalities have been employed.

Types of acne scar

- Ice-pick scar
- Atrophic macules
- Perifollicular elastolysis
- Perifollicular fibrosis
- Hypertrophic scars, Keloidal scars
- Rolling
- Box scars

Acne scars can be managed both medically & surgically. Medical management includes Tretinoin, hydroquinone, kojic acid and topical steroids. But they are of little value and are used only in treatment of post

inflammatory pigmentation. Hence surgical correction of acne scars is gaining importance nowadays.

Management of acne scars

1. Excision techniques like Punch excision, punch excision with skin grafting and punch elevation
2. Subcision
3. Dermabrasion
4. Microdermabrasion.
5. Laser
6. Fillers, and
7. Chemical Peeling

REVIEW OF LITERATURE

HISTORY

First description of acne in the Sushrut Samhita can be located under Kshudra Roga as Mukha Dushika. Acne existed in the Vedic kala (10,000 B.C to 500 B.C) and Samhita kala (200B.C to 400A.D) eras as Youvan Pidika.

According to these authorities, the word acne is a corrupt form of word Akme. The acne refers to youth or the prime period of the life[1]

EPIDEMIOLOGY

Acne vulgaris a universal skin disease affecting about 75 to 95% of adolescent population, 80% of affected individuals were between puberty and 30 years of age.

In India prevalence rate is about 50.60% in boys and 38.13% in girls, more commonly seen between 12 – 17 years of age[2].

DEFINITION

Acne is a chronic self – limiting inflammatory disease of the pilosebaceous units, seen mainly in adolescents, characterized by seborrhoea, presenting with pleomorphic variety of lesions, consisting of open and closed comedones, erythematous papules and pustules and in more severe cases nodules. In many cases a degree of scarring will ensue.[3]

ETIOPATHOGENESIS

Factors precipitating or aggravating acne

- Genetic influence has been accepted with very high concordance between monozygotic twins, in whom the sebum excretion rate (SER) is virtually the same. Persons with persistent acne have a strong positive family history [4-7].
- Cosmetics
- Repeated irritation
- Sweat – in about 15% of patients sweating reduces acne formation[8]
- Menstruation – premenstrual flare was noted in about 2 to 7 days before menstruation. This could be due to alteration in hydration of pilosebaceous epithelium [9]

- Stress – can induce acne. Acne induced stress and meddling of acne lesions aggravate appearance[10]

- Sunlight – natural sunlight improves acne which may be due to the cosmetic effects of tanning[11]

- Smoking – smoke contains polycyclic aromatic hydrocarbons and arachidonic acid, inducing phospholipase A2 dependent inflammatory pathway [12]. Smokers also consume diet containing high saturated fat and lower polyunsaturated, linoleic acid compared to those who do not smoke.

- Pregnancy – may aggravate or may cause remission

- Diet – dairy products, sweets, chocolates, fat. High glycemic diet results in hyperinsulinemia, triggering a cascade of endocrine factors like insulin like growth factor 1, alters signaling of retinoids, rise of androgen thus mediating acne formation [13].

- Drugs like topical or systemic steroids, anitconvulsants, anti depressants induce acne[14]

Pathogenesis of acne

1. Excess sebum production
2. Follicular epidermal hyperproliferation
3. Increased colonization of the pilosebaceous duct with Propionibacterium acnes
4. Inflammation and immune response.
5. Hormones

1. Increased sebum production

Sebaceous activity is regulated by androgens of gonadal or adrenal origin [15, 16]. The Dehydroepiandrosterone sulphate gets converted to dihydrotesterone and this binds to sebocyte resulting in excess sebum production and development of comedones in acne prone persons [17]. Androgens regulate function by binding to nuclear androgen receptors (ARs) present in the sebaceous gland.

Mechanism of excessive sebum production

1. Excessive androgen synthesis
2. Excess of free androgen which may be associated with relative low level of sex hormone binding globulin (SHBG)
3. An increased target response mediated by conversion of testosterone to more active dihydrotestosterone
4. Increased binding of androgen to its receptor

Androgen effects on the pilosebaceous unit are reasonably well documented. The variability in the response of this unit is not completely understood [18, 19]. Testosterone and DHT act through a single nuclear androgen receptor, with dihydrotestosterone (DHT) as the most active ligand.

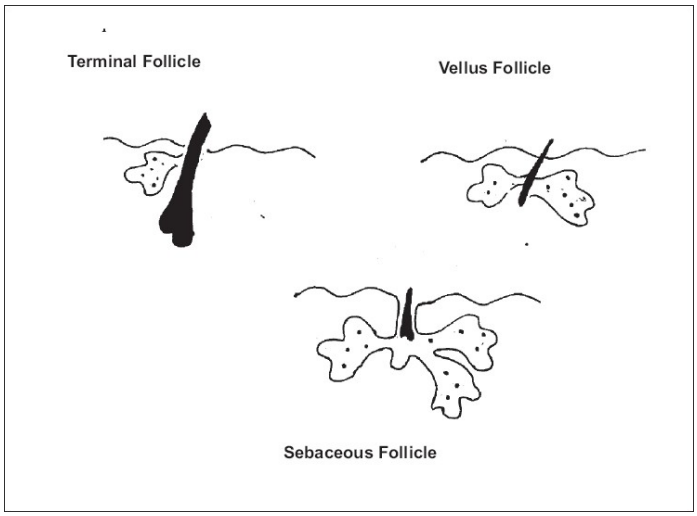
Acne does not occur simultaneously on all susceptible sites correlates as sebum excretion varies from follicle to follicle. In acne patients, there is marked variation in individual follicular sebum excretion. This hypothesizes that certain follicles may be prone to acne and that an enhanced peripheral (end organ) response to androgens is a probable factor [20]. Weak prohormones (DHEA, DHEAS and androstenedione) only act after conversion to more potent androgens testosterone and 5α-DHT.

Sebaceous glands in some areas show abnormally high 5α-reductase activity. In addition, abnormally high levels of plasma DHT and urinary 5α-androstanediols, considered to be biological markers of cutaneous androgen metabolism. This have been identified in some female acne patients[21]. Androgen action on the sebaceous gland may be independent of serum hormone levels. There are two forms of 5α-reductase. 5α-reductase type I is the most relevant in acne supported by the fact that Finasteride, an inhibitor of type II 5α-reductase, does not reduce sebum production and the fact that patients with a deficiency of

type II 5α-reductase have normal sebum levels. Regional differences in the activity of type I 5α-reductase in isolated sebaceous glands from various body sites also support the end-organ hyper-responsiveness theory for acne [22]

2. Alteration of follicular keratinization

Initial alteration of follicle is in the infundibular portion, it becomes hyperkeratotic with increased cohesion of keratinocytes. The increased proliferation of infundibular keratinocytes and their cohesiveness results formation of plug in the follicular ostium. Plug results in accumulation of keratin, sebum, and bacteria in the follicle, resulting in dilation of upper hair follicle, producing microcomedones[23].



3. Increased colonization with P.acnes bacteria

Propionibacterium acnes, is a diptheroid aerobic bacteria in nature. It is a commensal in pilosebaceous follicle. P.acnes and,

P.granulosum are the main species. They live in an environment along with Staph.epidermidis and M.furfur, the latter exerts control on the growth of P. acnes[24]. The cornified plug within the pilosebaceous duct obstructs sebum outflow, allowing propionibacteria to multiply and also preventing them and their products of metabolism from escaping onto the skin surface. P.acnes binds to toll like receptors 2(TLR2) inducing NF-kB switching leading to production of pro-inflammatory cytokines[25].

4. Inflammation and immune response

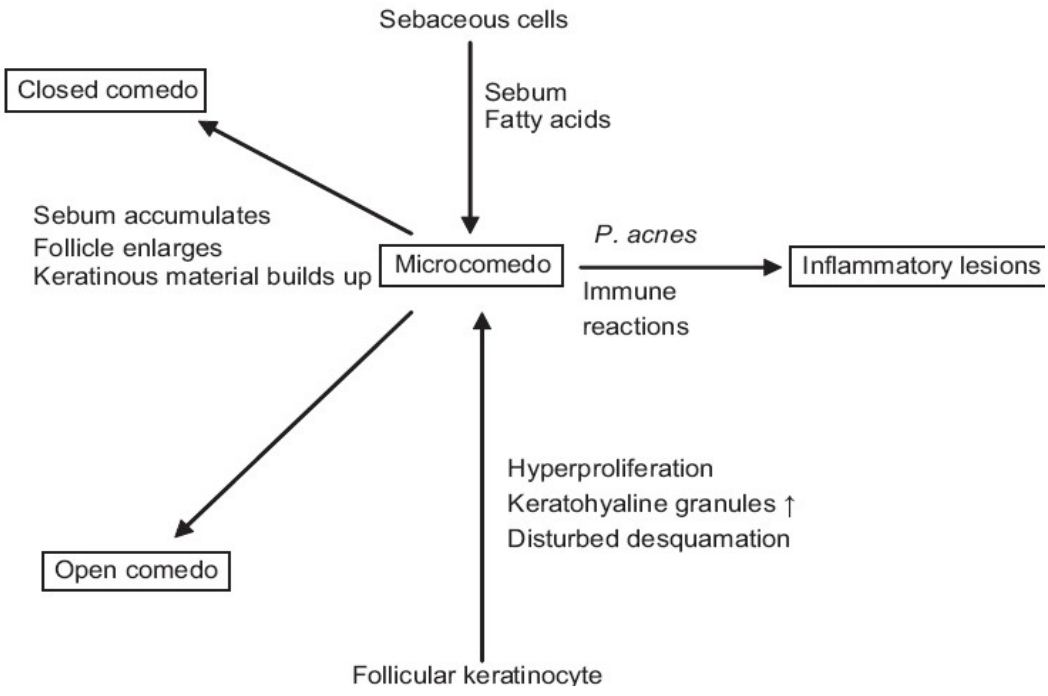
This is the major component for pigmentary disturbances and scarring. One hypothesis is that linoleic acid, which is known to be deficient in acne, could lead to an alteration in the integrity of the barrier function within individual follicles [26]. Basement membrane of the follicle wall remains intact in uninvolved follicles, a soluble antigen yet undiscovered could be a trigger for inflammation. Inflammation up regulates sebum production, in genetically predisposed individuals, elaborates IL-1 alpha initiating comedogenesis[27]. Sebum production is also influenced by neuropeptides, the reason for stress induced aggravation of acne.

Clinical features

The disease is clinically characterized by both inflammatory & non inflammatory lesions[28].

Non inflamed lesions of acne are called comedones.

- Black or open comedones size 0.5mm dm
- White or closed comedones
- Sand paper comedone – multiple very small white-heads, common over forehead and gives rough, grity feel on touch.
- Submarine comedones – 0.5 cm dm, resides deep in skin creases and is the cause of recurrent nodular lesions
- Secondary comedones – seen after exposure to dioxins pomades, steroids and other drugs.
- Microcomedo gives rise to inflammatory lesions in acne
- Non inflammatory lesions develops into superficial and deep lesions are papules and pustules,
- Deeper lesions more commonly seen in males; extends in depth and also involve large areas.
- Sinus formation may be seen between nodules and deep pustules, resulting in disfiguring cosmetic effects and inevitable scarring[29].


Sebaceous cells
Sebum
Fatty acids
Closed comedo
Sebum accumulates
Follicle enlarges
Keratinous material builds up
Microcomedo
P. acnes
Immune
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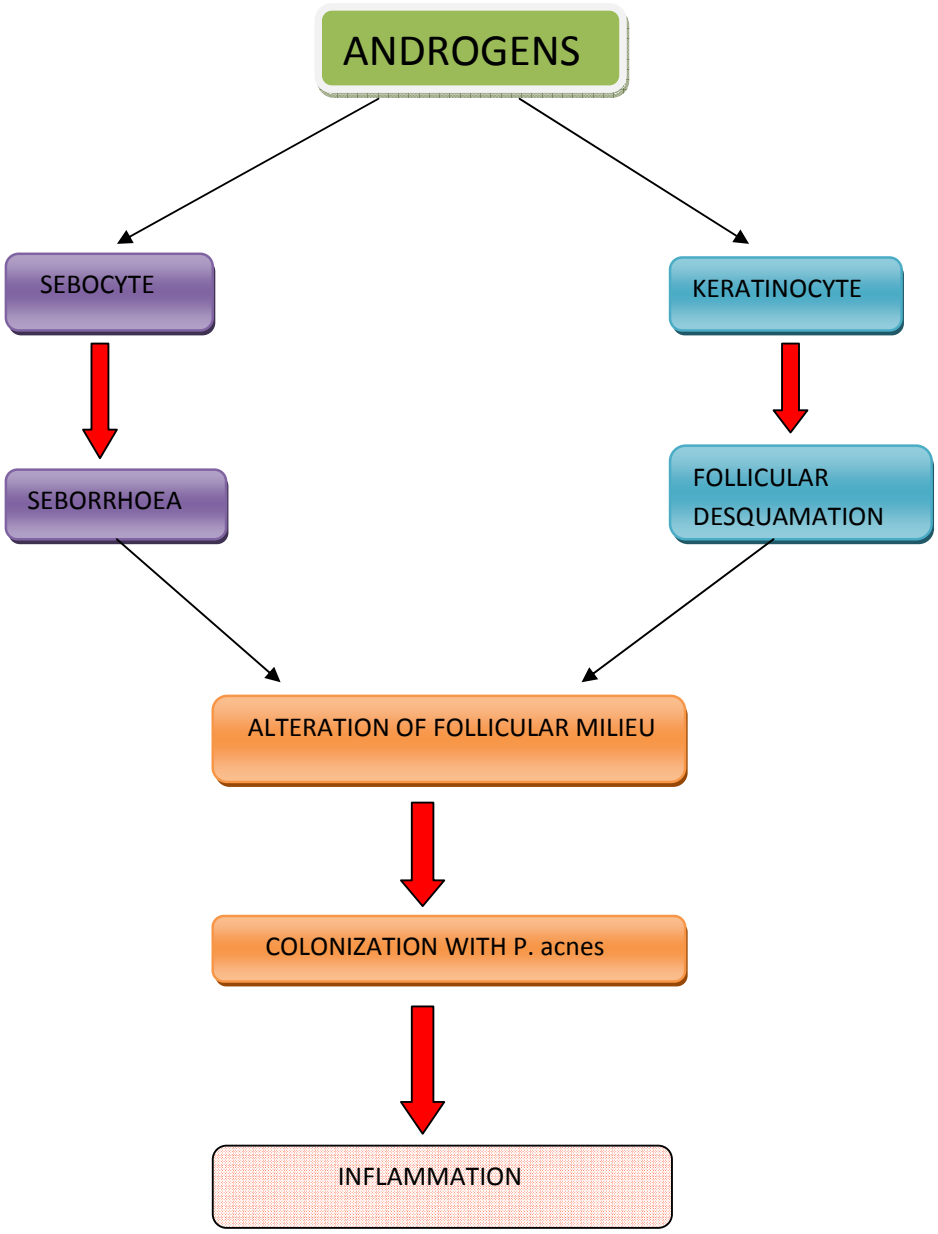
Inflammatory lesions
Hyperproliferation
Keratohyaline granules ↑
Disturbed desquamation
Open comedo
Follicular keratinocyte


ANDROGENS
SEBOCYTE
KERATINOCYTE
SEBORRHOEA
FOLLICULAR DESQUAMATION
ALTERATION OF FOLLICULAR MILIEU
COLONIZATION WITH P. acnes
INFLAMMATION

Pathogenesis of scar formation

Risk of scarring differs between individual patients with acne and not dependent on severity of the disease[30].

CMI is involved in inflammatory lesions of acne, also helps in clearance of antigen and tissue damage. The differences in cell-mediated immune responses in developing and resolving inflamed lesions between acne patients who were prone to scarring, and those with the same degree of inflamed acne not prone to develop scarring. Scarring in acne is determined by severity of inflammation as measured by depth and duration. In lesions from acne patients who were vulnerable to scar, a predominantly adaptive immune response was present, which was persistent and up-regulated in resolving lesions. The number of CD4 T cells was approximately half of those found in lesions of non-scarrers, but a high percentage of these cells were skin homing memory/effector cells, suggesting that these patients were sensitized to the causative antigens[31]. In developing lesions, although the numbers of macrophages, blood vessels and vascular adhesion molecules were high and similar to those present in lesions of non-scarrers, the numbers of Langerhans' cells and the level of cellular activation were low, and comparable to levels found in normal skin, indicative of an ineffective response. However, in resolving lesions, there was an up-regulation of the response with greater cellular activation and a further influx of macrophages and skin homing

memory/effector cells. Certainly, the strong macrophage presence represents a dominant force in this response. Thus, it may be interpreted that, in patients prone to scarring, there is a chronic delayed-type hypersensitivity reaction provoked by a persistent antigen which these patients are initially unable to eliminate[32].

Keloidal scar occur on dark skin and is mapped to chromosome 2q23 and 7p11[33]. Damage to epidermis resulting in erythema and pigmentation is reversible, while damage to dermis resulting in atrophic scars is partially reversible and is irreversible in hypertrophic scars[34].

Remodelling of collagen the final step in tissue repair, is mediated by matrix metalloproteinases (MMPs), causes the damage, and tissue inhibitors of metalloproteases (TIMPs) reduces the damage[31-37].

Alteration of ratio of MMPs/TIMPs i.e when it is minimal atrophic scars occurs, when the ratio is more hypertrophic scars occurs.

Types of acne scars

- Atrophic – icepick, boxcar, macular atrophic, Perifollicular elastolysis, rolling
- Hypertrophic – keloidal, popular, Perifollicular fibrosis
- Mixed
- Unclassified

Classification of acne scars Goodman and Baron [38]

Grade 1: Macular - Erythematous, hyperpigmented, or hypopigmented marks

Grade 2: Mild Disease (Mild atrophy)

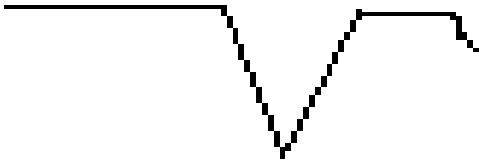
Grade 3: Moderate Disease (Moderate scarring)

Grade 4: Severe Disease - Scarring not flattened with manual stretching of the skin

In acne patients not prone to scarring, the time course was typical of a type IV delayed hypersensitivity response. In developing lesions there was significant angiogenesis and vascular adhesion molecule expression, with a large influx of activated CD4 T cells, macrophages (CD68) and Langerhans' cells (CD1a)[39]. Cell recruitment peaked at 48 h, after which there was a decrease in leukocytes, cellular activation and a return to normal levels of blood vessels and vascular adhesion molecules in resolving lesions. Of the CD4 T cells, 50% were skin homing memory effector cells (CD45RO,CLA) and naive cells (CD45RA) cells, whilst the remainder were unclassified (CD45RO−, CD45RA−, CLA−), which suggests that effective resolution occurred by both non-specific/innate and adaptive immune mechanisms[40].

Morphological description of different types of acne scars

- Ice-pick scars – jagged, firm atrophic scars, most evident on the cheeks. arises from comedones alone.



- Rolling scars are shallow and wider than 4–5mm, and they appear as superficial shadowing and rolling or undulating appearance to the overlying skin.

- Boxcar scars are round to oval depressions with sharply demarcated vertical edges

- Perifollicular electrolysis (PFE) is commonly found on the trunk which are multiple, follicular and atrophic

- Keloidal scars extend beyond the sites of original inflammation and are most prevalent on the trunk.

- Hypertrophic scars do not extend beyond the extent of the original inflammation

Treatment of acne scars

Acne scar procedures grouped by procedure type[41]

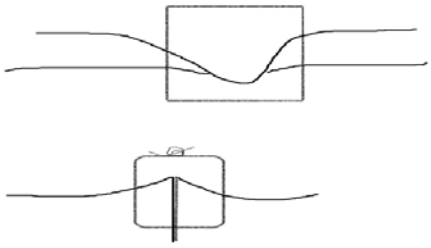
Resurfacing	Lifting	Excision	Others
Chemical peels full face, Chemical Reconstruction Of Skin Scars (CROSS)	Subcision	Punch excision Elliptical excision	Skin needling
Dermabrasion ❖ LASER resurfacing ❖ Ablative/non ablative ❖ Fractional	Fillers ❖ Directly under scars ❖ Voluminizing ❖ Autologus fat transfer ❖ Punch elevation	Punch grafting	Facelift Combination techniques

For atrophic scars

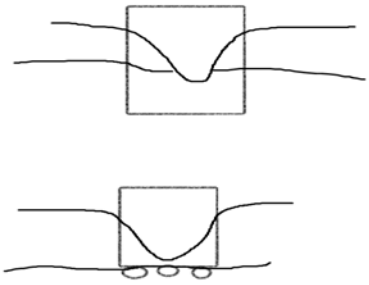
Scar excision

Tiny /minute, well defined scars can be removed by punch excision, elevation, subscision.

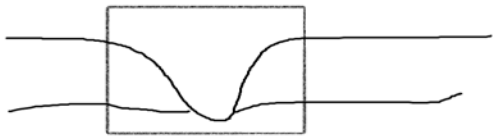
- Punch excision - The scar is excised with a punch and the skin is sutured together

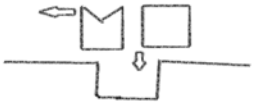


- Punch elevation punch of appropriate size is inserted in to the scar till base and adhesions at sides are cut, the plug is then raised. This procedure is called punch elevation

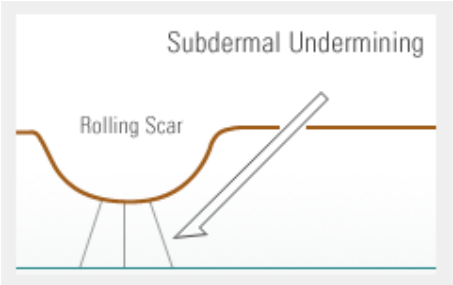


- Punch excision and grafting scars are removed with a punch and is replaced by donor graft from retroauricular area





- Subcision - surgically freeing up the deeper layers of the skin so the scars are no longer bound down. A needle or surgical scissors are used to physically separate the dermis and subcutaneous tissue.



- Microdermabrasion – Skin resurfacing with Aluminium oxide or sodium chloride microcrystals to superficially remove stratum corneum and epidermis. Microcrystals circulate through high speed system and get collected in a waste container.

- Chemical peeling – exfoliation of skin induced by the use of chemical cauterant or escharotics agent on the skin

Peels used commonly

- ❖ TCA
- ❖ Alpha hydroxyl acids -glycolic acid
- ❖ Salicylic acid
- ❖ Retinoic acid
- ❖ Jessner's solution
- ❖ Phenol
- ❖ 5 – Fluorouracil
- ❖ Alpha keto acids

Types of Chemical peels

- ❖ Very superficial- glycolic acid 10 to 30%, TCA 10%, Jessner's solution
- ❖ Superficial- glycolic 50 to 70%,TCA 10 to 35%
- ❖ Medium depth – glycolic >70%, TCA 35 to 50%
- ❖ Deep – phenol 88%

- TCA CROSS –focal application of higher concentrations of trichloroacetic acid (TCA) and is pressed hard on the entire

depressed area of atrophic acne scars. This technique is called chemical reconstruction of skin scars (CROSS)

- Laser – CO2 Laser is used it has a wavelength of 10600 nm. Resurfacing of acne scars done by sculpturing the edges.

- Fillers

Purified brine collagen is injected in the defective areas.

Now degradable, synthetic implants like hyaluronic acid were used, to avoid risks of sensitizations & foreign body reaction.

In acne scars – freeze dried, radiation treated fascia lata from human cadaver implant is placed at site of each scar by creating an intradermal pocket at site with help a needle.

Alternate method is autologus fat transplantation to acne scars

Selection of procedures according to types of scars

1. Ice-pick scars – punch excision, CROSS chemical peels
2. Rolling scars – subcision, fillers, Fractional Laser, skin needling
3. Boxcar scars – CROSS chemical peels, punch elevation, fractional LASER, skin needling, focal dermabrasion, punch excision, elliptical excision

4. Keloidal and hypertrophic scars – potent topical steroids, intralesional Triamcinolone, cryotherapy and silicone gel application

Microneedling/ Percutaneous collagen induction

The first to notice the advantages of skin needling were Orentreich et al. They were the first to introduce the term Subcision from the contraction of the term ‘‘Subcutaneous Incinsionless’’ surgery. It is a method of cutting under a depressed scar, wrinkle or contour using a tri-beveled hypodermic needle inserted under the skin through a needle puncture, adjacent to the scar[44-48].

The procedure attempts to raise the base of the defect to the level of the surrounding skin surface by 2 distinct mechanisms:

- The procedure of surgically freeing the skin from its adhesion to deeper tissues. This results in skin elevation

- The introduction of a controlled trauma initiates wound healing with resultant production of connective tissue that elevates the depressed scar.

The technique involves 3 different methods:

- Inserting and withdrawing movement of the needle under the scar, in a linear and simple fashion.

- Horizontal movement of the needle under the scar like fanning

- The needle is moved vertically underneath the scar

Needling of the skin has been in use since 1995 to achieve percutaneous collagen induction (PCI) [49]. It is an effective method in treating acne scars of grades 2–3 . The technique involves needling the skin multiple times with a small needle to induce collagen growth.

Camirand and Doucet treated scars with a tattoo gun to 'needle abrade' them although this technique could be used on extensive areas, it was laboriously slow, and the holes in the epidermis were too close and too shallow. All these techniques worked because the needles break senile collagen strands in the most superficial layer of the dermis that tether the scars or wrinkles. It is assumed that this process promotes removal of damaged collagen growth and induces synthesis of new collagen immediately under the epidermis [50-55].

The principle of using dermaroller modality is to provide collagen induction therapy. This is done by causing a minute injury to the dermis with use of microneedles. This results in starting of the wound healing cascade and hence utilizing the body's own wound healing mechanisms. Skin reacts to the intrusion of micro-needles like any other skin penetrating object. In microneedling the needles are so fine and thin that

tissue damage is very minimal. The skin integrity actually stays intact and only the body's own production of collagen is stimulated[56]

Each tiny wound goes through the three classic phases of wound healing

- **Inflammation**
- **Proliferation**
- **Tissue remodeling**

The needle only penetrates through the epidermis and does not remove it, so the epidermis is only pierced and will rapidly heal in a matter of hours, since keratinocytes are highly elastic and flexible[57]. This injury, minute as it might seem, does cause some localized damage and micro-bleeding, not visible on the skin. The needle pricks of the instrument will penetrate to their full length in to the dermis and initiate the inflammation phase. Rupture of skin capillaries lead to extravasation of blood cells and serum into the surrounding tissue. This results in formation of hundreds of minute clots which increases the vascular permeability, the chemo attraction for leukocytes and the recruitment of fibroblasts in the wounded area. Platelets cause clotting and release chemotactic factors like Platelet Derived Growth Factor (PDGF), Transforming Growth Factor (TGF) and Fibroblast Growth Factor (FGF) that initiate an invasion of other platelets, leucocytes and fibroblasts.

Neutrophils act on the ‘‘damaged’’ tissue and removes the debris of the old and damaged collagen and microclots. This reaction is automatic and produces a surge of activity that inevitably leads to the fibroblast being directed to produce more collagen and more elastin[58-60].

The re-epithelialisation occurs within a few hours after needling, and is because of keratinocyte migration rather than proliferation. When the keratinocytes accumulate together, they starts producing all the components to re-establish the basement membrane with laminin and collagen types IV and VII.

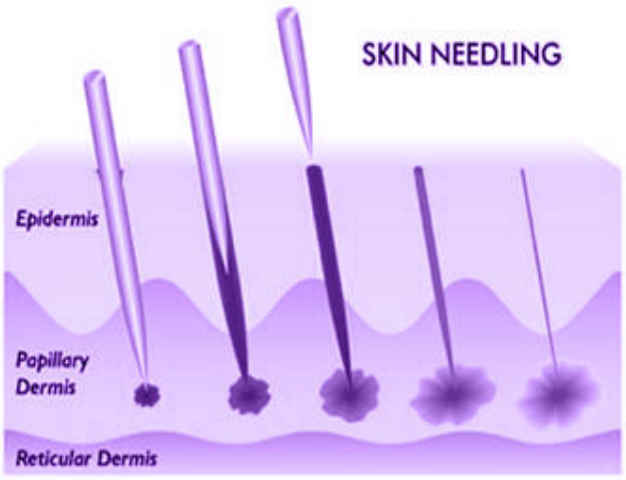
One or days after the injury, the keratinocytes start proliferating and thicken the epidermis. Fibroblasts migrate into the wound and produce collagen, proteoglycans, elastin and other matrix proteins. Initially after needle damage, the disruption of the blood vessels causes a moderate amount of hypoxia. The low oxygen tension stimulates the fibroblast to produce more TGF, PDGF and VEGF (Vascular Endothelial Factor). Procollagen mRNA is also upregulated but this cannot cause collagen formation because oxygen is required and that only occurs when re-vascularisation occurs.

Revascularization occurs quite soon after needling. TGF is a powerful chemotactic agent for fibroblasts, which migrate into the wound at about 48 hours after injury and start producing collagen I and III, elastin, glycosaminoglycans and proteoglycans. Collagen type III is the

dominant form of collagen in the early wound healing phase and becomes maximal by five to seven days after injury. The collagen is laid down on the upper dermis just below the basal layer of the epidermis.

Tissue remodelling continues for months after the injury and is mainly overtaken by the fibroblasts. By day 5 after the session, the fibronectin matrix is laid down along the axis in which fibroblasts are aligned and is the same axis that collagen will also lay down. TGF and other growth factors play an important role in the formation of this matrix.

Combination treatment with subcision, punch elevation, Fractional laser, peeling gives excellent results.



Advantages

- Well tolerated well accepted by the patients
- Cost-effective compared with Laser therapies
- Can be done on all skin types
- Done on areas not suitable for peeling or laser resurfacing, such as near eyes
- Can go back to work the very next day
- Easy to use
- Power independent
- Less time consuming

Disadvantages

- cannot be used in other type of acne scar

Dermabrasion

Sequential planning of the skin to desired level using an electrical or manual dermabrader.

Principle

The goal of dermabrasion is the remodelling and restructuring of the papillary dermis and upper reticular dermis without injury to the deep reticular dermis. The principle behind is that injury above the level of deep reticular dermis heals without scarring[61-67]

Sequential levels of dermabrasion

- Pigmented skin removed – epidermis
- Appearance of large number of tiny pin-point bleeding points – superficial papillary dermis is reached
- Appearance of fewer number of larger bleeding points with whitish grey ridging of fine collagen denotes junction of deep papillary dermis and upper reticular dermis
- Firmer surface with increased large bleeding and breaks in parallel lines and ridges – junction of upper and mid reticular dermis, maximum level of dermabrasion
- Herniation of fat with yellow appearance – subcutaneous fat, signs of over abrading

Advantages

- Cost effective
- Short recovery compared to LASERS
- Less complications compared to LASERS and other procedures

Disadvantages

- Patient discomfort
- Requires surgical skill
- Larger areas – need general anaesthesia, longer procedure time
- Prolonged downtime

AIM AND OBJECTIVES OF THE STUDY

1. **To compare the effectiveness of percutaneous collagen induction therapy versus dermabrasion in post acne scars**

2. **To look for the adverse effects during and after the procedure**

MATERIALS AND METHODS

Study design

- Type of study – non randomized prospective comparative study

Study population

- Sample size – thirty patients divided into two groups

Study period

- May 2011 to October 2012(18 months)

Group A – Microneedling (n=15)

Group B – Dermabrasion (n=15)

Study analysis

- Chi square test using spsf software

Place of study

Department of Dermatology,

Government Stanley Medical College & Hospital

Chennai

From about 100 patients with acne scar attending our OP, thirty patients were selected according to the inclusion and exclusion criteria. They were questioned about the duration of the problem, any previous treatment taken for the same and any history of keloidal tendency. They all were examined for any new acne lesions, keloidal and hypertrophic scars. Out of the thirty patients 20 had rolling type of acne scars, 4 had boxcar scars, 3 had both and 3 had ice pick scars. They were given pre-treatment consultation, information sheet about the surgical procedure and fully explained about the pros and cons of the procedures. After their full understanding and willingness only they were enrolled in the study. Pre-operative assessment and investigations were done

Pre treatment consultation

- Patients were well informed about the method of the procedure, course of the treatment and possible adverse effects.
- Informed and written consent were obtained
- Complete history regarding onset, duration, any other co-existing systemic illness, past history of any treatment for the same, Isotretinoin use, immunosuppressive agents, Hepatitis B co-infection were also noted. Any H/O previous herpetic lesions also asked for.
- Counseling of the patient is utmost important

- The motivation of the patient is judged
- Explain the nature and downplay the degree of improvement
- Discuss with the patient about side effects and pigmentary changes and also about the time taken for recovery of normal skin

Pre-surgery assessment

- A thorough clinical examination as given in ANNEXURE-I was done.
- Patient were also examined for any keloidal tendency

Pre-operative workup

- HB, TC, DC, ESR, Platelet count
- Bleeding time, clotting time
- Fasting blood glucose
- Renal function test – blood urea, serum creatinine
- SGOT
- SGPT
- Screening for Hepatitis B, HIV, VDRL testing.
- Informed consent and photographs

INCLUSION CRITERIA

- Both sexes
- Age 15- 35 years
- Acne scars – rolling, boxcar, ice – pick scars
- Patient willing for follow up and to take photographs

EXCLUSION CRITERIA

- Pregnant or lactating women
- Scars other than acne, other types of acne scars
- Patients having keloidal tendency
- Active skin infections, active acne
- H/O Herpes labialis
- Bleeding diathesis

Study procedure

Group A - Percutaneous collagen induction/ microneedling

Priming (atleast 2 weeks prior)

- The patients were advised to apply topical vitamin A or vitamin C

Instruments required

- Dermaroller - a cylindrical roller studded with 192 tiny fine needles arranged in rows of eight, 0.5 to 1.5 mm in length and 0.1 mm in dm, for acne scars needle length should be 1mm
- Cap, mask, eye pad for the patients
- Sterile gauzes
- Sterile saline soaked gauzes

Procedure

- The area to be treated is anaesthetized with topical anasethesia like EMLA
- After about waiting period of 45 minutes, the area is wiped clean with surgical spirit
- The area to be treated is stretched with one hand
- The hand piece of the instrument is grasped tightly with other hand, and procedure is started
- Rolling is done in various directions horizontal then vertical and oblique directions

- The appearance of minute pin point bleeding points or petechiae is characteristic.
- Entire procedure is completed in about 15 to 20 minutes
- No dressing needed for the area
- Sunscreens with a high SPF applied immediately after treatment

How to sterilize the instrument

- The microneedling instrument storage container, filled with surgical spirit in such a way that the roller head is submerged.
- After 10 to 15 minutes disinfection is complete
- The container is emptied and hot water is poured in it to remove leftover alcohol and microbes
- The dermaroller and the container have to dry completely. The dermaroller instrument is placed inside the container and lid is closed air tight.
- Outside the container the patient's name, identification marks and the number of sittings were written.

Post Procedure advice

- Photoprotection for a week
- Patient is advised to wash the face with cool water for the first 48 hours after treatment and not to rub the area
- Advised not to apply conventional make-up products for about 12 hours after treatment
- As the microholes close immediately, postoperative infections do not occur.
- Patient reviewed after one week then weekly thereafter
- Procedure repeated once in six weeks depending on the response

Group B – Dermabrasion

- Priming (atleast 2 to6 weeks prior)
- Sunscreens – to prevent post operative hyperpigmentation
- Tretinoin – for faster post-operative healing stopped 3 days prior to surgery

Instruments required

- Electrical dermabrader or hand engine 10,000 – 25000 rpm
- Diamond fraizes
- Wire brushes
- Cap, mask, gown, gloves, saline soaked sterile gauze pieces

Procedure

- Informed consent
- The area to be treated is anaesthetized with topical anasethesia like EMLA
- After about waiting period of 45 minutes, the area is wiped clean with surgical spirit
- The scars to be treated were marked and the area to be abraded is outlined with marking ink
- The skin is stretched with one hand
- The hand piece of the electrical dermabrader is grasped firmly and is performed in a direction perpendicular to the axis of rotation of the diamond fraize.

- The dependent area is abraded first then proceeded upwards in order to avoid obscuring the field by blood

- First there was appearance of large number of tiny pin bleeding points , then fewer number of larger bleeding points with whitish grey ridging of fine collagen, abraded further till the appearance of firmer surface with increased large bleeding points and breaks in parallel lines

- Individual scars were dermabraded with pear shaped fraize

- Complete hemostasis achieved by applying ice cold saline sponges

- Area is cleansed thoroughly with normal saline to remove remnants of epidermis

- Dressing done with non adhering chlorhexidine gauze

- Post operative antibiotics, analgesics were prescribed

Post operative follow up

- Dressing was changed after one week

- Crusting was seen in about 7 to 10 days

- The patients were reviewed every week to look for the response, and for any complications

- Procedure repeated after 6 weeks depending on the response

- Patient advised to avoid sun exposure.

OBSERVATION AND RESULTS

Total number of patients in the study: 30

Study population

This study includes 30 patiens 20 male and 10 female patients

Youngest age is 16 years of age and oldest is 32 years of age. All fulfilled the inclusion and exclusion criteria

Table 1 – Age wise distribution of study population

Age group	Number of patients
15 to 20	12
21 to 25	9
26 to 30	7
31 to 35	2

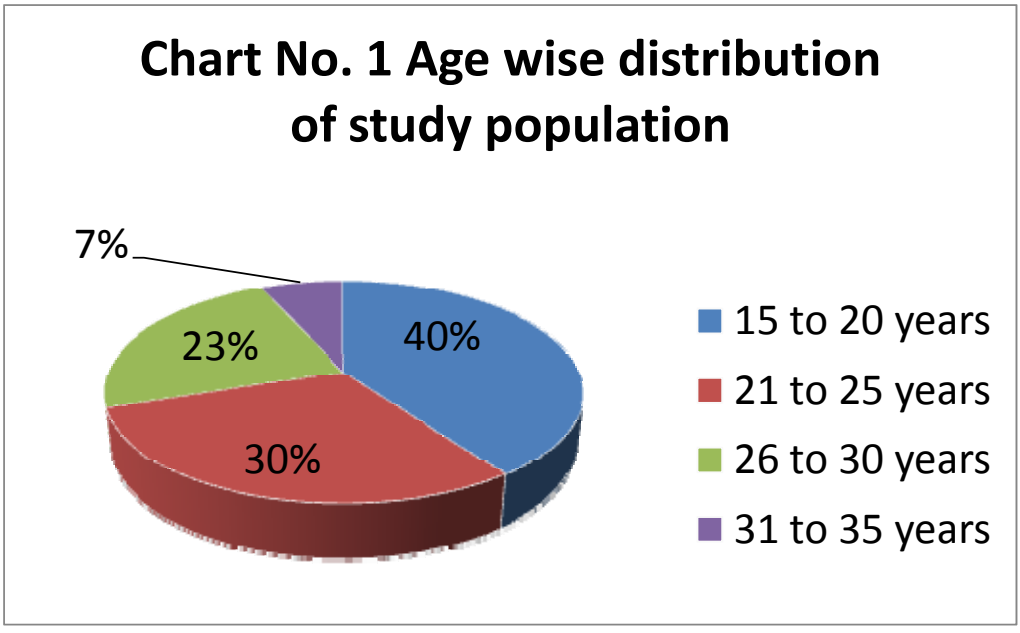
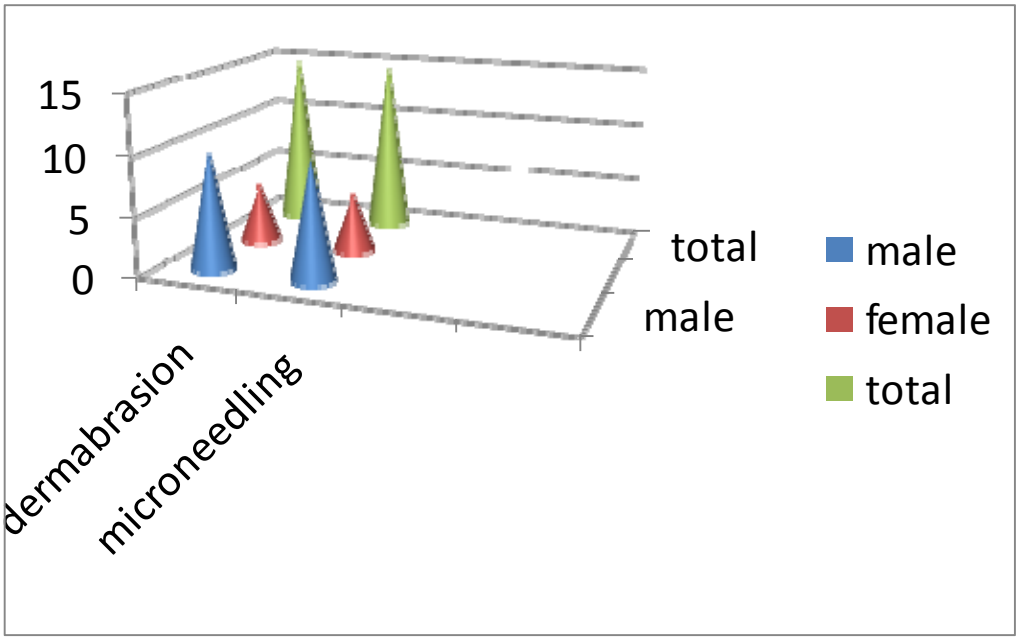


Chart No. 1 Age wise distribution of study population
7%
40%
23%
30%
15 to 20 years
21 to 25 years
26 to 30 years
31 to 35 years

Table 2: Sex wise distribution of study group

Name of procedure	Males	Females	Total
Percutaneous collagen induction	10	5	15
Dermabrasion	10	5	15

Chart No : 2 Sex wise distribution of cases



Of the 30 acne scar patients included in the study fifteen of them had rolling scars, eight had boxcar scars, five of them had ice-pick scars, and two of them had both rolling and boxcar scars

In dermabrasion approximately 80% of patients were males and in microneedling 67% of them were males.

Table 3: Type of acne scars included in the study

Type of scar	Number of patients
Ice pick scar	5
Rolling	15
Boxcar scar	8
Both boxcar and rolling	2

Chart No 3: Number of patients in different types of scar

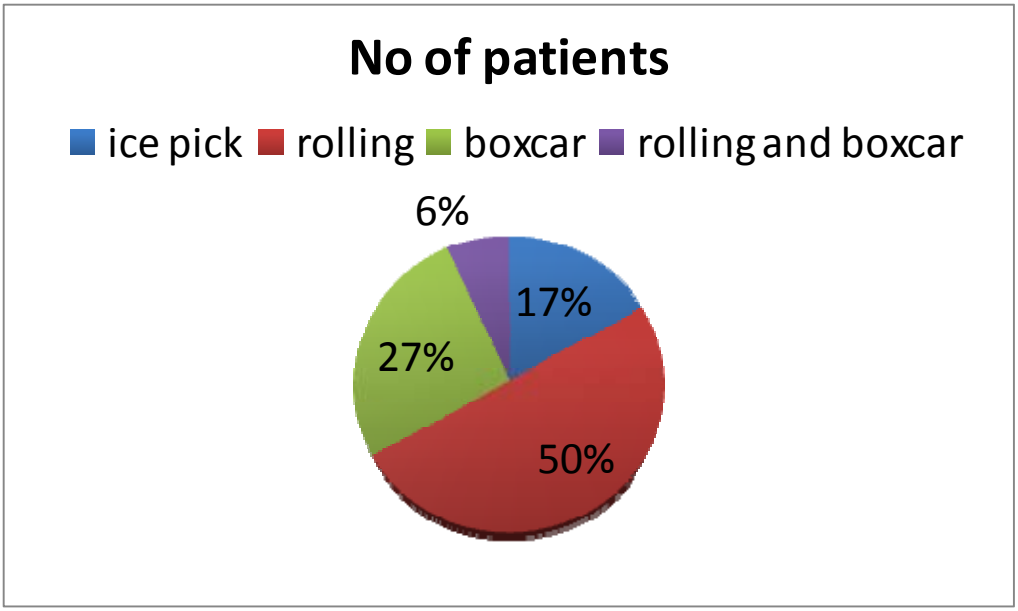
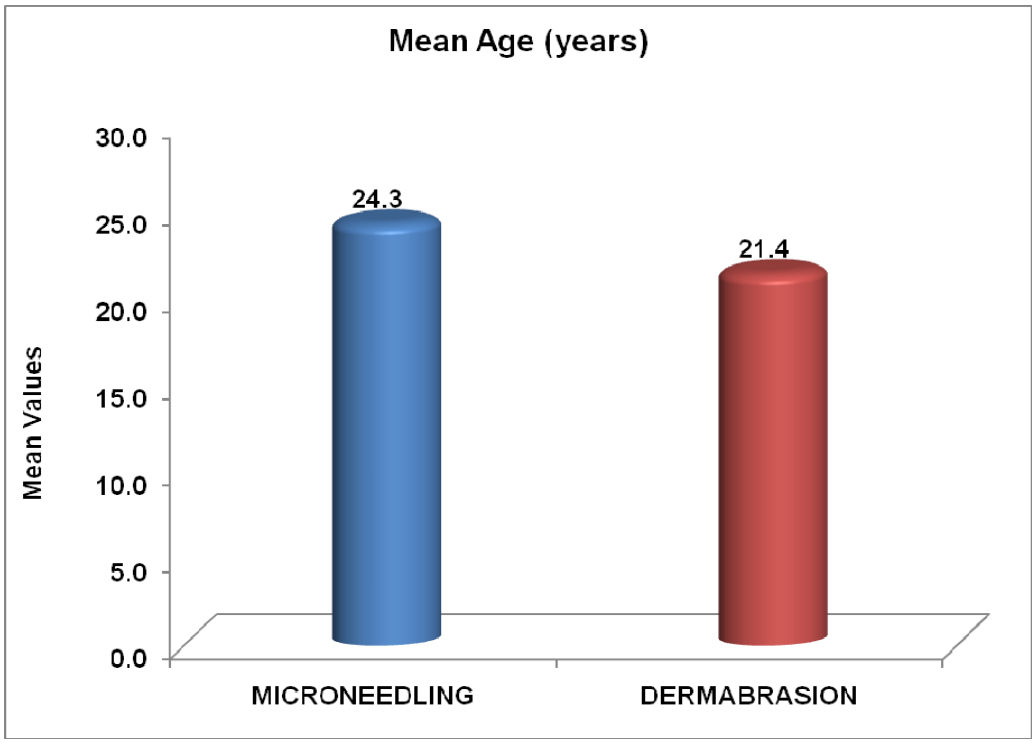


Chart No 4: Mean age distribution in the study



The mean age of patients in microneedling is 24.3 years, and in dermabrasion are 21.

GROUP A – MICRONEEDLING / PERCUTANEOUS COLLAGEN INDUCTION

Out of the fifteen patients included in the study only one patient experienced severe erythema which persisted for more than twenty-four hours, then subsided in about 8 to 72 hours.

Five of them experienced moderately severe erythema which subsided in about six hours; four of them experienced only mild erythema.

Rest of them did not experience any adverse events. Edema was present in almost all the patients which subsided in about 48 hours.

Pigmentation was seen only in about five patients, and they were given depigmenting agents, along with sunscreens

Almost all the patients experienced little or no pain after the procedure. It has a very little downtime – a few hours of tingling and redness observed in many.

Total number of sittings required varied between patients. Two of the patients improved with first sitting, about 60% improvement, three of them improved in second sitting, seven improved after the fourth sitting. Interval between each sitting was about 6 weeks

Table 5: Percentage of improvement and number of sittings

Number of Sittings Required	Number of Patients	% of Improvement (average)
1	2	50 TO 60%
2	3	50 TO 60%
3	1	50 TO 60%
4	5	40 TO 50%
4	2	30 TO 40%
4	2	< 25%

Percentage of improvement was measured by both subjectively and objectively.

Table 6: Scar Improvement Response with Microneedling

% of Improvement	Grading	Number of Patients
< 25%	Poor	2
25 To 50%	Moderate	7
50 To 75%	Marked	6
>75%	Excellent	None

GROUP B – DERMABRASION

Out of the 15 patients, 9 of them experienced erythema over the abraded area which persisted for about ten days. There was no report of infection from any of the patient in group B, as they were given prophylactic systemic antibiotics for about 5 days post operative.

Dressing was removed after one week. Crusting took place in about 7 days for five patients, 9 days in 9 patients and 12 days in one patient.

Pigmentary changes were seen in almost all the patients. Mild in six patients, moderate in nine patients.

Total number of sittings of dermabrasion differed between individuals

Post procedure edema was seen in about seven patients

Other adverse effects like infections and milia were not encountered in this study.

Table 4: Complications in Dermabrasion

Complications	Number of patients
Dyschromia	15
Erythema	9
Edema	7
Milia	Nil
Infections	Nil

Chart No 5: Percentage of Complications In Group B

(Dermabrasion)

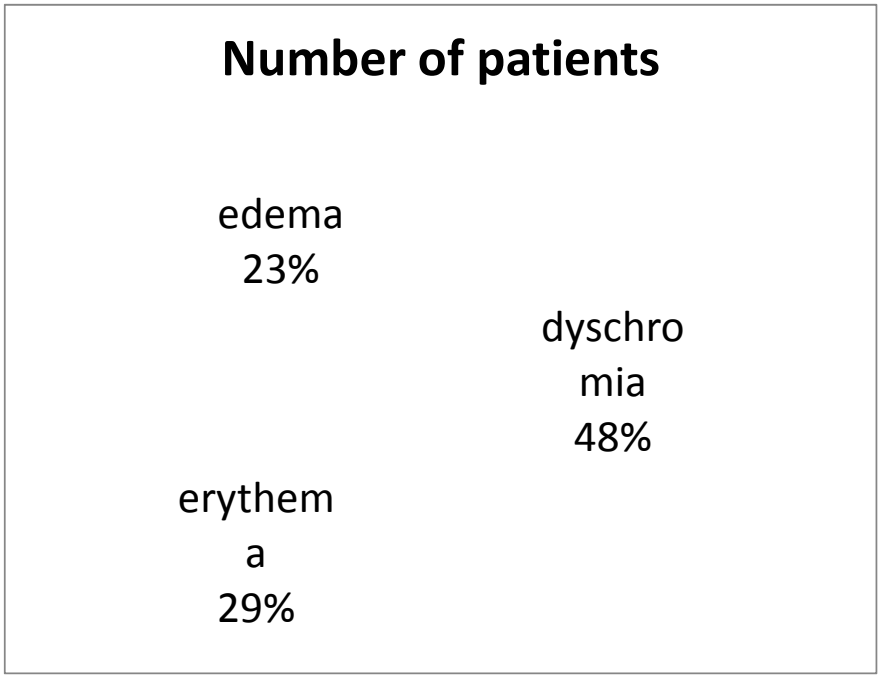


Table 7: Chi-Square test to compare the proportions between groups

Variables		Group				Total		P-Value
		Micro-Needling		Dermabrasion				
		N	%	N	%	N	%	
Gender	Male	10	66.7	12	80.0	22	73.3	0.682[a]
	Female	5	33.3	3	20.0	8	26.7	
Types of Scar	Rolling	8	53.3	7	46.7	15	50.0	0.661[a]
	Boxcar	5	33.3	3	20.0	8	26.7	
	Ice pick	2	13.3	3	20.0	5	16.7	
	Boxcar, Rolling	0	0.0	2	13.3	2	6.7	
Quartile grading scale	Grade 1, <25%	3	20.0	8	53.3	11	36.7	0.041[b]
	Grade 2, 26–50%	6	40.0	5	33.3	11	36.7	
	Grade 3, 51–75%	6	40.0	2	13.3	8	26.7	
Dyschromia	Negative	9	60.0	0	0.0	9	30.0	0.001[a]
	Positive	6	40.0	15	100.0	21	70.0	
Erythema	Negative	4	26.7	6	40.0	10	33.3	0.439
	Positive	11	73.3	9	60.0	20	66.7	

The significance of this study shows that p value is much significant in microneedling with regard to improvement as measured with quartile grading scale.

Pigmentary disturbances more commonly seen in dermabrasion, as noted by the p value which is significant.

Table 8: Independent samples t-test to compare mean values between groups

Variables	Group	N	Mean	Std. Dev	P-Value
Age (years)	Microneedling	15	24.27	3.615	0.099
	Dermabrasion	15	21.40	5.409	
DLQI - Before	Microneedling	15	10.80	1.265	0.884
	Dermabrasion	15	10.73	1.223	
DLQI - After	Microneedling	15	5.87	1.407	0.001
	Dermabrasion	15	7.80	1.568	
DLQI difference	Microneedling	15	4.93	1.486	0.003
	Dermabrasion	15	2.93	1.870	

The p value of DLQI of both procedures were significant but is better in microneedling

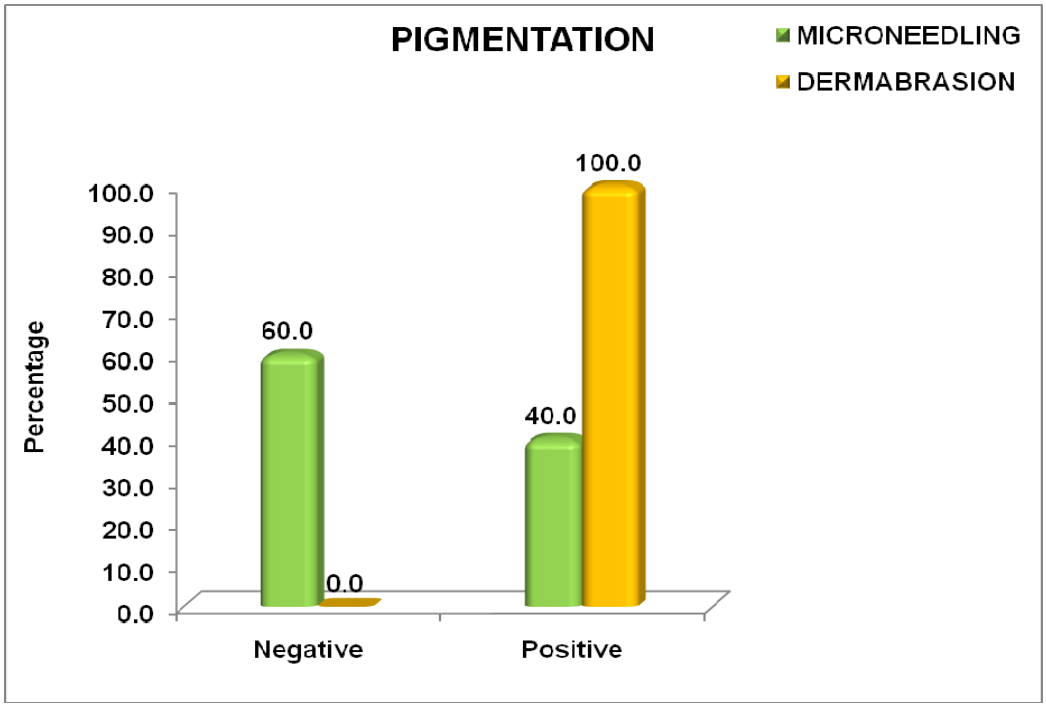
Table 9: Paired Samples t-test to compare mean values between Before and After

Group		N	Mean	Std. Dev	P-Value
Microneedling	DLQI - Before	15	10.80	1.265	<0.001
	DLQI - After	15	5.87	1.407	
Dermabrasion	DLQI - Before	15	10.73	1.223	<0.001
	DLQI - After	15	7.80	1.568	

This data suggests that both the procedures have high significance within the same group.

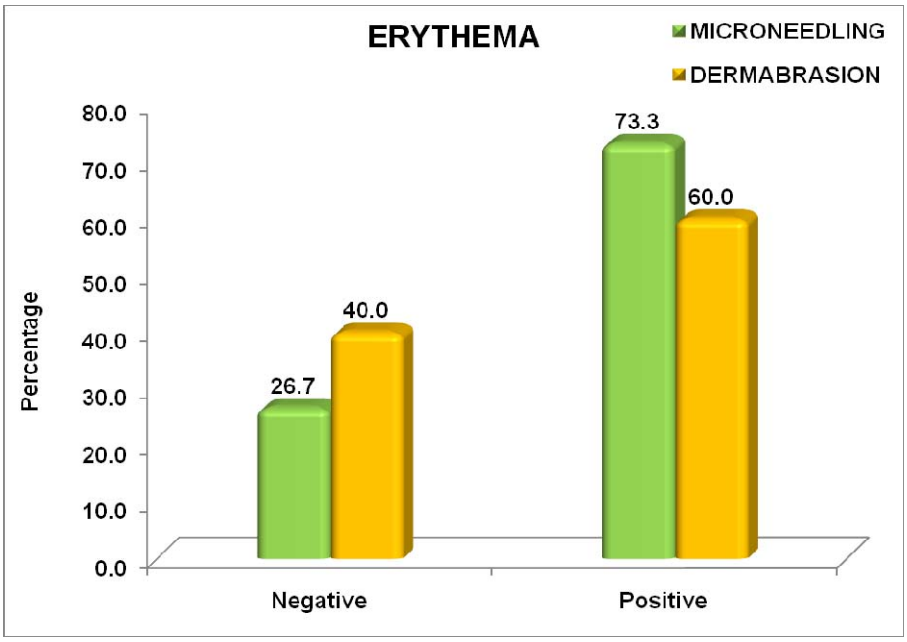
The sign test for paired data ($P < 0.05$), highlighted that the median of the differences is negative, showing that the reduction in severity grade of acne scars, before and after microneedling, should be considered significant.

Chart No 6: Percentage of pigmentary changes noted in both Groups



This comparative study is significant as the p value is less than 0.001 for dyschromia.

Chart No 7: Percentage of Population in both Groups with Erythema



Post procedure erythema was seen in about 73% in group A and 60% in group B.

Chart No 8: Improvement in Quartile Grading Scale in both Groups

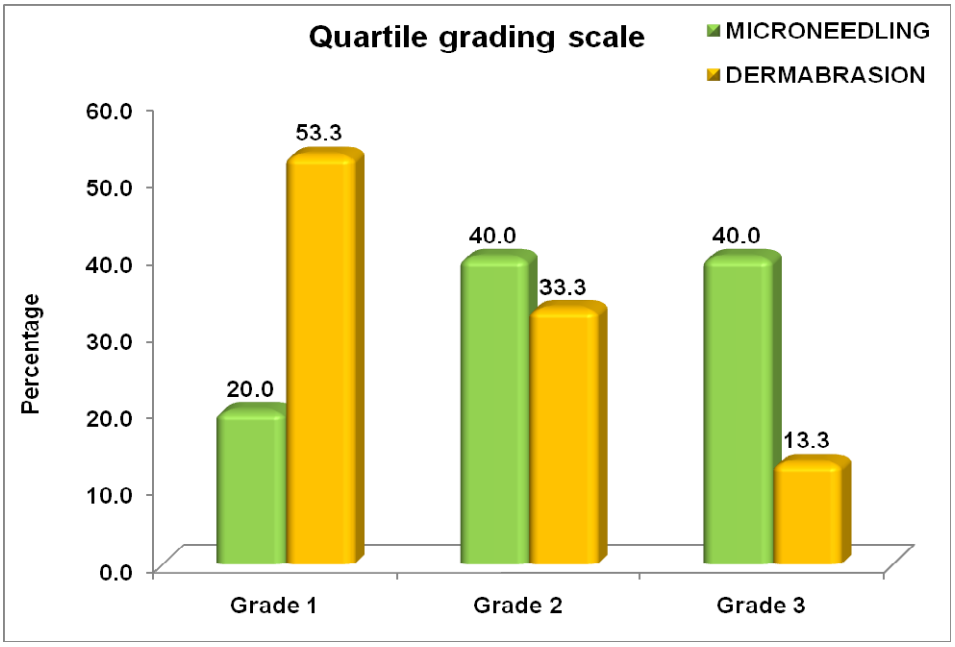
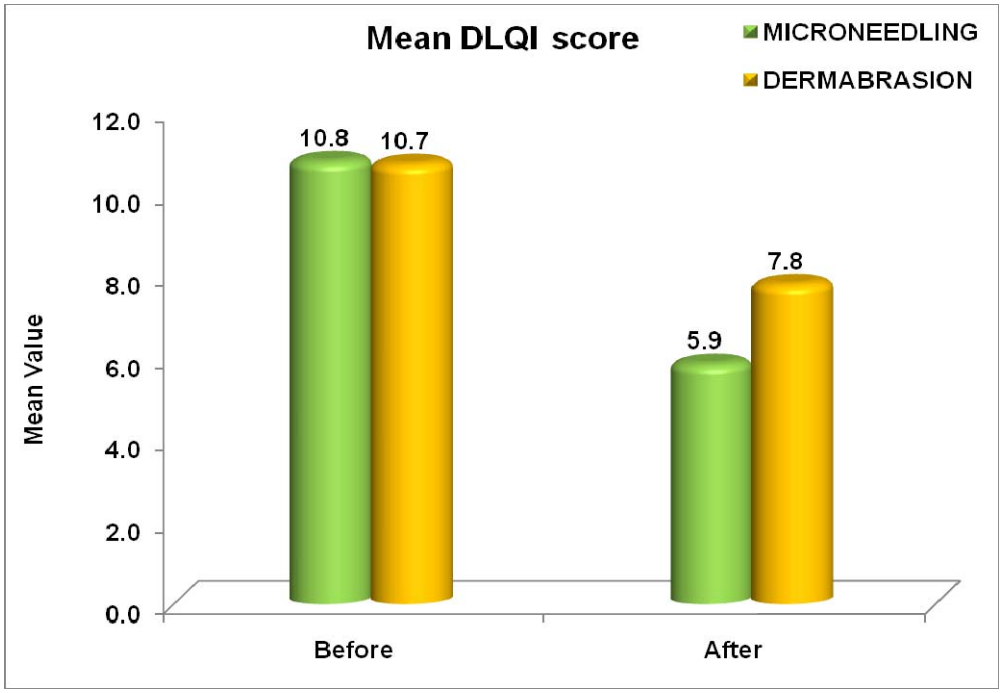
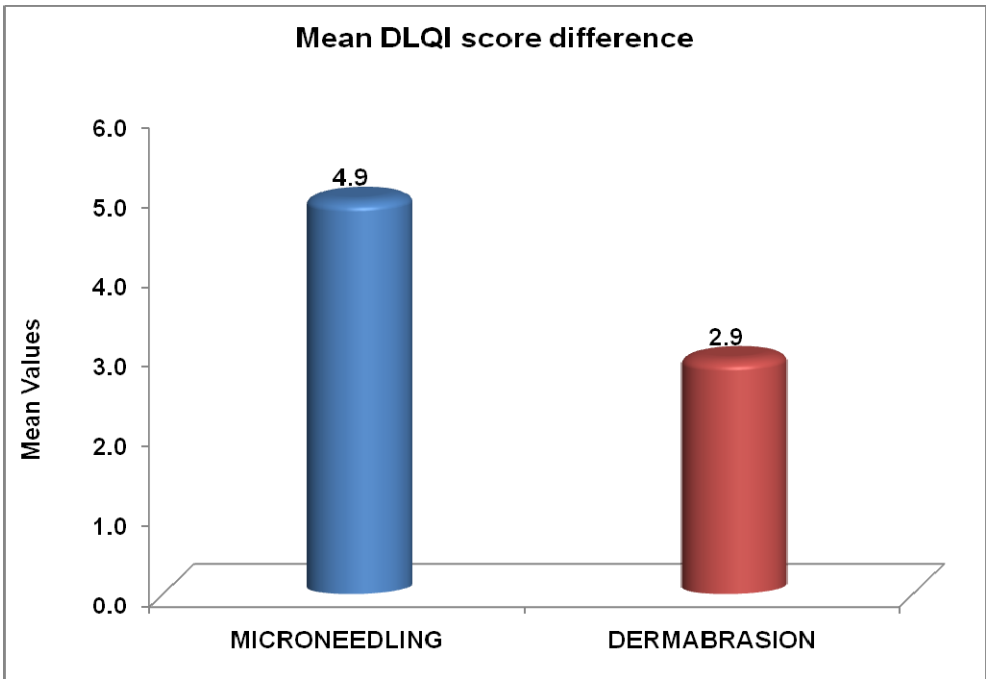


Chart No 9: Impovement in DLQI in Both Groups




Mean DLQI score difference
Mean Values
6.0
5.0
4.0
3.0
2.0
1.0
0.0
4.9
2.9
MICRONEEDLING
DERMABRASION

MICRONEEDLING PHOTOS

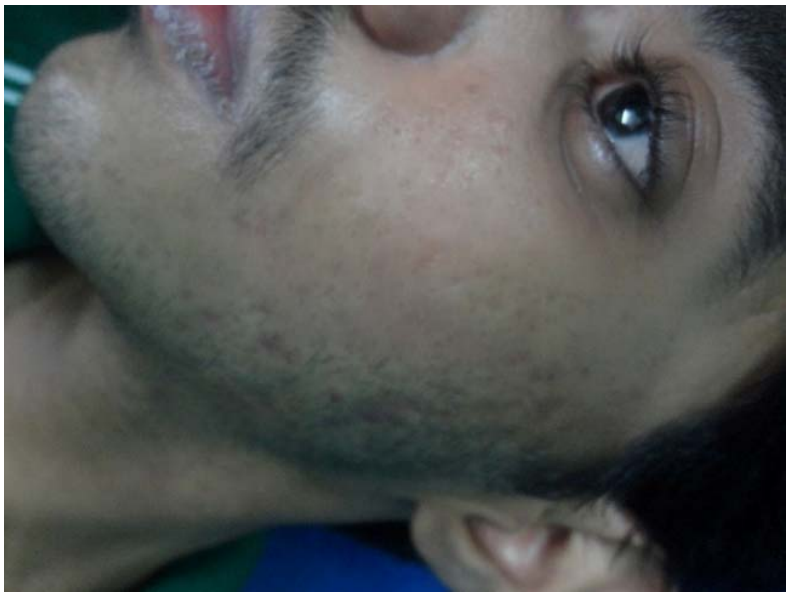
Procedure of Microneedling

Before Microneedling

Immediate Post Operative

After 2nd Sitting Significant Improvement

Before



4 weeks after 1st sitting marked improvement

Before

After 2nd sitting garde 3 improvement

Before

After 3rd sitting

Procedure of Dermabrasion

Before dermabrasion

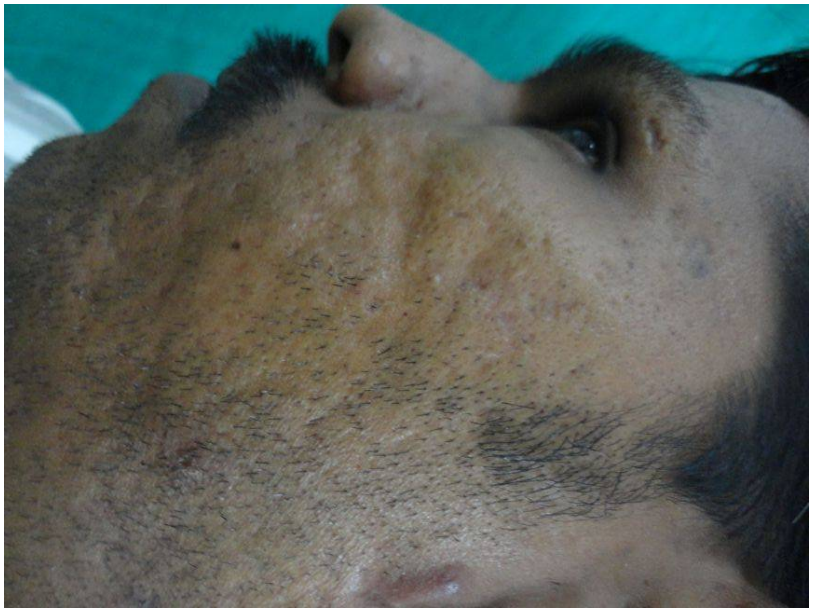
Six weeks after dermabrasion 1st sitting

Before dermabrasion

Six weeks after 1st sitting grade 2 improvement

Before dermabrasion

Treatment failure even after 4 sittings of dermabrasion

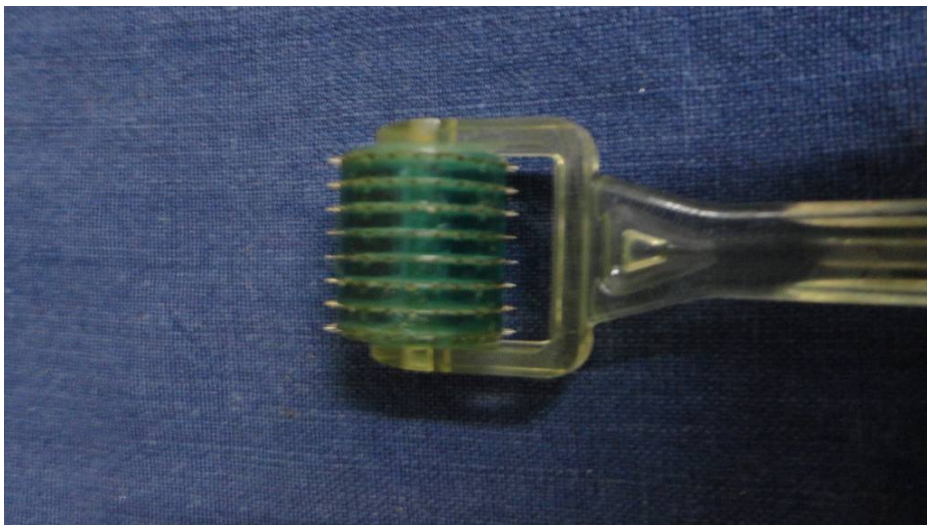


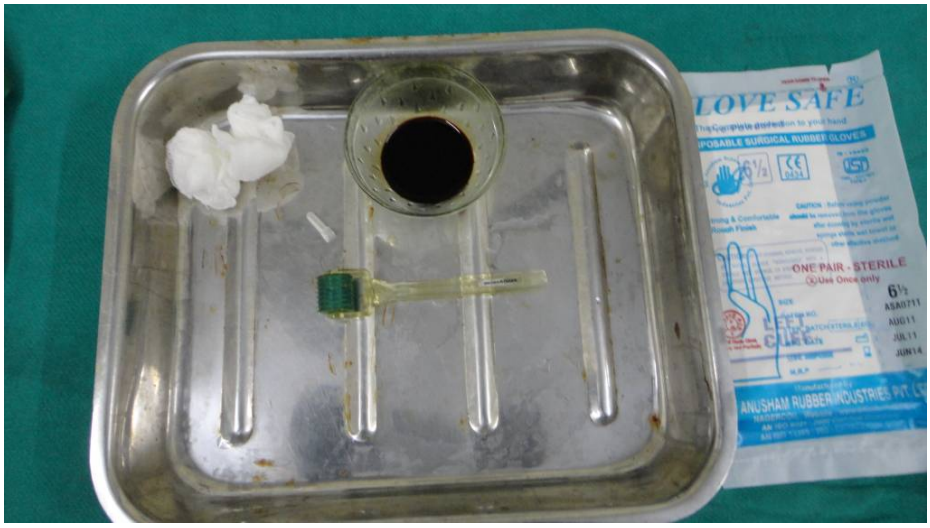
Before dermabrasion

Pigmentary disturbances in dermabrasion

DERMABRASION INSTRUMENT

MICRONEEDLING INSTRUMENT





DISCUSSION

It is better to prevent than to treat. Preventing the formation of scars in acne reduces the morbidity associated with acne scars. People with Acne scars may face physical, aesthetic, psychological, and social consequences that may be associated with substantial emotional and financial costs.

Microneedling

Microneedling provides collagen induction through controlled mechanical stimulation of the dermis resulting in softening of scars leading to realignment of senile collagen bundles. There is complete preservation of the epidermis during the procedure, also there is no permanent damage to the skin. Not associated with pain during procedure no bleeding, infection, discoloration or other complications on the treated area. Healing takes place within 24-48 hours. The very next day patient can go back to his routine. This procedure needs only 3-5 days protection from ultraviolet light.

In Group A patients who underwent microneedling the success rate was 70 to 80%

There is significant improvement in the DLQI score in microneedling which was reduced to a mean of 5 from a score of 10. Improvement of quartile grading scale was also noted in about 70 to 80%

In a study from Srinagar[68] which showed excellent response in about 70 to 80% for atrophic acne scars with microneedling therapy which correlated with our study.

In a study conducted in Maharashtra[69] Microneedling alone or when combined with other modalities showed a significant degree of improvement about 70 to 80% improvements in rolling scars. The study was conducted in about thirty five patients with rolling scars significant improvement was seen in grade 2 and 3 scars.

Complication was not that much with microneedling only erythema was seen in about 73% that too subsided in about 2 to 3 days. Pigmentary changes were seen in about 40% which also subsided in about 3 weeks.

Dermabrasion

Dermabrasion (DA) consists of sequential planing of the depressed scars with electrical and/ or manual abraders and allowing the wound to heal by secondary intention, so as to achieve a leveling effect to make the scar less conspicuous.

In our study the success rate of dermabrasion was about 40 to 50%. The patients who responded well had rolling and boxcar type of scars. There is also a reduction in the DLQI score from a mean of 10 to about 7.

In a study by SS Savant[70] for acne scars using dermabrasion 40 to 50% % of success rate was noted this result correlates with our study.

Regarding the complications Pigmentary disturbances seen in almost all the patients and erythema was seen in about 66%

Other modalities of treatment

LASERS which has been used for resurfacing also yields positive results the only drawback is the cost.

Chemical peeling also showed good results in treatment of acne scars, advantage is that is cheap, done as outpatient procedure. Disadvantage is that post inflammatory hypo pigmentation may be seen in some.

In a study by George J. Hruza, subcision gave better results of 51 to 75% in boxcar rolling type of scars with no complications [73].

In a study from New Delhi trichloroacetic acid CROSS (chemical reconstruction of skin scars) is better in rolling scars [71,72].

In a study from Egypt TCA CROSS was better than Nd : YAG laser severity of acne scars were greatly reduced in the TCA CROSS study group[74]

There is no comparative studies published till date between effectiveness of microneedling and dermabrasion in post acne scars

Only individual studies are available the results of these studies correlated with our study.

CONCLUSION

- To conclude percutaneous collagen induction(PCI) therapy is easy to perform provides good results with minimal adverse effects.
- Percutaneous collagen induction is also safe, least expensive and most effective in treating superficial scars.
- The patient feels better after the procedure, and they could perform their daily routine without any difficulty
- It has only minimal side effects like erythema and edema which subsided in about six hours maximum in 2 to 3 days
- Microneedling is cost effective aptly described as a poor man's LASER
- Microneedling is less painful unlike dermabrasion
- Microneedling can be done as an outpatient procedure unlike dermabrasion in which sometimes general anesthesia may be needed.
- In dermabrasion the patients were unable to do their daily routine because of the raw area created by the procedure

- Dermabrasion, even though cost effective the patient compliance is poor, tolerability of the patient is also poor. Does not give a cosmetic appearance after the procedure

- It also has prolonged downtime minimum of 4 to 5 weeks for complete healing

- Dyschromia is almost seen in all the patients which is a disadvantage of dermabrasion, deeper dermabrasion results in milia or scarring

- Power driven dermabrader requires expertise

- To conclude both the procedure are cost effective and gives good results. The results of PCI were better when compared to Dermabrasion

- Skin needling is a simple and rapid method for safe treatment of acne scars and a suitable procedure for various dermatological conditions.

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GOVERNMENT STANLEY MEDICAL COLLEGE HOSPITAL
DEPARTMENT OF DERMATOLOGY
ACNE SCAR REVISION

NAME: **AGE:** **SEX:**

OCCUPATION:

ADDRESS:

D.O.A **D.O.D**

OP.NO: **IP.NO:**

Chief Complaints: H/O scar.

Relevant history: H/ O trauma, spontaneous onset,

H/ O drug intake

H/ O previous surgeries

H/ O chicken pox,

H/ O bleeding diathesis, smoking, alcoholism.

General examination: Pulse rate

BP: _____mm/hg

Dermatological examination: pigmentation, scar, keloid, active infection. Depth of scar

Investigation: Hb% TC,DC, Platelet count, ESR.

Blood sugar, urea, creatinine, electrolytes

Liver function tests

Urine albumin, sugar, deposit

Bleeding time, clotting time

ICTC, VDRL

Diagnosis: **Post Acne Scar**

CONSENT FORM

Mr. /Mrs./Miss
Age
Address
Phone no
Name of the procedure

I _____, give my free and full consent to Dr._____ for the undergoing microneedling/ dermabrasion for the acne scars , the nature and consequences of which have been explained to me. I have been completely explained the procedure, the need for investigations, its results and possible side effects in my regional language. I understand that more than one session may be needed for obtaining results. I understand the limitations of the procedure and also the final results.

I also give my consent that during the procedure if any complications arises, I may be given an emergency treatment best suitable to me without asking my prior permission

I have been provided adequate opportunity to seek information or withdraw from the study anytime during the study.

_____

Signature of the patient/ guardian.

_____

Signature of doctor.

_____

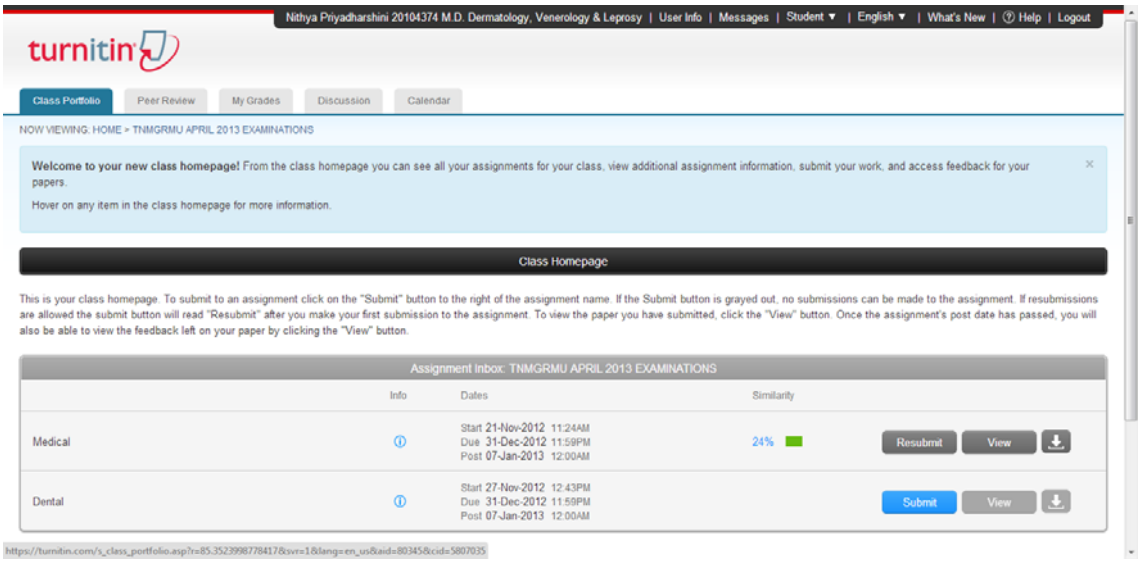
Signature of witness.

MASTER CHART

MICRONEEDLING

No.	Name	Age/Sex	Type of Scar	DLQI		Quartile grading scale	Complications		
				Before	After		Pigmentation	Erythema	Worsening
1.	VINOD	17/M	ROLLING	10	5	3	-	++	_
2.	NAGARAJ	26/M	BOXCAR	12	7	2	+	+++	_
3.	LAKSHMI	31/F	ROLLING	11	4	3	-	++	_
4.	AVINASH	21/M	BOXCAR	9	4	2	+	+	_
5.	ARAVIND	19/M	ROLLING	13	7	3	-	-	_
6.	DHIVYA	28/F	ROLLING	12	7	2	+	-	_
7.	PRAKASH	25/M	BOXCAR	10	5	2	-	-	_
8.	RAJA	22/M	ROLLING	12	5	3	-	+	_
9.	GEETHA	25/F	ROLLING	11	7	3	-	++	-
10.	JEGAN	26/M	BOXCAR	11	6	3	+	+	_
11.	KANNAN	24/M	ROLLING	11	8	1	--	+	_
12.	JOHN	27/M	BOXCAR	9	6	2	-	+	_
13.	SATHISH	22/M	ICE PICK	10	8	1	+	-	_
14.	SARANYA	24/F	ROLLING	12	5	2	-	++	_
15.	GOMATHI	27/F	ICE PICK	9	4	1	+	++	_

No.	Name	Age/Sex	Type of Scar	DLQI		Quartile grading scale	Complications		
				Before	After		Pigmentation	Erythema	Worsening
1.	NARESH	17/M	ROLLING	11	9	2	++	+	_
2.	GOWTHAM	17/M	ROLLING	13	7	2	++	+	_
3.	SURESH	30/M	BOXCAR, ROLLING	11	5	2	++	+	_
4.	SHANTHI	24/F	ICE PICK	10	8	1	++	++	_
5.	YASIR	17/M	BOXCAR, ROLLING	10	7	1	++	-	_
6.	ARCHANA	16/M	ROLLING	11	9	1	++	-	_
7.	CHARAN	19/M	BOXCAR	13	9	2	+	+	_
8.	NAVEEN	20/M	ROLLING	11	10	1	+	-	_
9.	RAMESH	19/M	ROLLING	12	9	2	+	+	-
10.	PRIYA	32/F	BOXCAR	10	5	3	++	+	_
11.	KADAR	18/M	ROLLING	9	9	1	++	_	_
12.	KUMAR	25/M	BOXCAR	11	6	3	++	_	_
13.	FAHID	18/M	ICE PICK	10	8	1	+	_	_
14.	DEEPA	19/F	ROLLING	10	9	1	+	+	_
15.	ANAND	30/M	ICE PICK	9	7	1	+	+	_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